Case Report

Psychological Autopsy and Necropsy of an Unusual Case of Suicide by Intravenous Toluene

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ABSTRACT

Toluene (methylbenzene; volatile hydrocarbon) is an industrial solvent that causes major injury to the lungs; the organ being the first capillary bed encountered. We report an unusual case of suicide by a 24-year-old male, paramedical professional, with fatal outcome within 16 h of intentional, intravenous self-administration of toluene, with clinical presentation of acute respiratory distress syndrome. Psychological autopsy revealed severe depressive disorder and solvent (inhalant) abuse, with marital disharmony as the precipitating stressor for suicide. Necropsy revealed diffuse congestion of internal organs like lungs and liver, epicardial petechial hemorrhages, and gastric hemorrhages. Treatment of toluene poisoning includes supportive care as no specific antidote is available. Early and aggressive management may be conducive to a favorable outcome with minimal residual pulmonary sequelae. Relevant literature of toluene poisoning was identified via PubMed, PubChem, ToxNet, Hazardous Substances Data Bank (HSDB), Embase, and PsycINFO. To our knowledge, this is the first case of suicide by intravenous administration of toluene in the literature.

Key words: *Fatal, suicide, inhalant- use disorder, intravenous injection, Toluene*

INTRODUCTION

Toluene (methylbenzene), an aromatic hydrocarbon is a non-corrosive, colorless, volatile, inflammable liquid with a strong pleasant odor. It is commonly used as an additive and solvent in numerous industries for automotive coatings, thinners, reactive diluent, rubber, adhesives, printing-ink, shoe polish, and correction-fluid diluents.[1] Unusual methods of suicide by intravenous administration of solvents like kerosene[2] and gasoline[3] are rare. We report an unusual case of suicide by intravenous injection of toluene. Relevant literature was searched via PubMed, PubChem, ToxNet, Hazardous Substances Data Bank (HSDB), Embase, and PsycINFO using keywords "toluene", "suicide", and "intravenous". To our knowledge, this is the first case of suicide by intravenous administration of toluene in the literature.

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CASE REPORT

A 24-year-old male with right-hand dominance, paramedical professional, presented to the Emergency Department at 0200 hours with chest discomfort, breathlessness, and hemoptysis, following intentional intravenous self-administration of 'correction-fluid diluent' (5 ml) at 0130 hours [Figure 1a]. The warning displayed on the 'diluent' bottle carton read: "Contains toluene. Do not drink or inhale. Misuse by drinking or inhalation can be harmful, even fatal" [Figure 1b]. On admission, Glasgow Coma Scale (GCS) score was E4V5M6, with blood pressure of 130/80 mmHg, pulse

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rate of 172 bpm, and oxygen saturation (SpO_2) of 77%. Pupils were 3 mm in size, round, regular, and reactive to light. Fundoscopy showed bilateral normal optic discs. Tachypnea with coarse crepitation in bilateral infrascapular and infraaxillary areas were noted suggestive of chemical pneumonitis. Neuromuscular examination was normal. Local examination revealed two puncture marks at the left cubital fossa indicating self-administration of toluene intravenously [Figure 1c].

Hematological investigations revealed raised hemoglobin (18 g/dL), leukocytosis (33,850/mm^3), normal platelet count, normocytic, and normochromic peripheral blood picture. Renal profile showed raised serum creatinine (1.7 mg/dL), normal blood urea (39 mg/dL) and serum electrolytes. Liver profile revealed increase in total bilirubin (2.67 mg/dL), direct bilirubin (1.11 mg/dL), indirect bilirubin (1.52 mg/dL), serum glutamate ortho-transaminase (68 U/L), with other enzymes and proteins within normal limits. His random blood glucose was 215 mg/dL, possibly due to stress-induced hyperglycemia. Electrocardiogram showed ventricular tachycardia with 170-180 bpm. His chest roentgenogram showed bilateral, diffuse haziness suggestive of pneumonitis. Management included conservative treatment with intravenous hydration, antibiotics, pantoprazole, ondansetron, and mucolytics. At 0930 hours, patient had abdominal pain, hematemesis with deterioration of SpO_2 to 75% with oxygen therapy. Due to acute respiratory failure, mechanical ventilation was initiated. Endotracheal aspirate was hemorrhagic in nature. At 1500 hours, patient was in state of stupor with GCS score of 4 (E1cV1tM2). At 1730 hours, patient had sudden cardiac arrest following ventricular fibrillation. Cardiopulmonary resuscitation including defibrillator efforts were in vain and patient was declared as dead at 1810 hours same day (16 h after intravenous toluene administration). Psychological autopsy revealed pervasive depression, apathy, insomnia, anorexia, and reduced social interactions for the past 4 months, with marital discord and 'sniffing' since 2 years. He was diagnosed as severe major depressive disorder with possible inhalant use disorder as per Diagnostic and Statistical Manual of Mental Disorders (DSM)-5 criteria.[4] Necropsy revealed grossly edematous and congested lungs [Figure 2a], epicardial petechial hemorrhages [Figure 2b], hepatic congestion, gastric mucosal congestion and petechial hemorrhages with contents of 50 ml blackish-red colored blood [Figure 2c], and pale intestines. Two puncture marks were noted over the left cubital fossa with no remarkable local skin changes [Figure 1c]. Two puncture marks of intravenous cannula and test-dose (antibiotics) were noted in right forearm. Examination of brain and kidneys was unremarkable. Histopathological findings were suggestive of chronic distal renal tubular acidosis (type-II). Gas chromatography of the blood revealed toluene.

DISCUSSION

Toluene is absorbed into circulation by inhalation, ingestion, and dermal or eye contact.[1] Being a hydrocarbon of low viscosity, it has a direct toxic effect on lung tissue — interfering with gas exchange and leading to interstitial pulmonary edema and severe hypoxemia caused by impaired diffusion.[3] It is one of the commonly abused inhalant.[5] Inhaling low to moderate doses of toluene can cause tiredness, confusion, ataxia, amnesia, nausea, anorexia, and loss of hearing and color vision that usually disappear when exposure is stopped. Inhaling high doses of toluene in a short-time may cause cardiovascular effects (tachycardia, hypo/hypertension, dysrhythmia), pulmonary effects (acute pulmonary edema, asphyxia, aspiration/chemical pneumonitis, respiratory depression, apnea), and central nervous system effects (tremors, ataxia, euphoria, hallucinations, convulsions, delirium, coma, death). Fatal dose of toluene by inhalation is 1,800-2,000 ppm or with blood levels of 25 mg/L [Table 1].[1,6-9] Ingestion of toluene may cause irritation of the digestive tract with nausea, vomiting, and abdominal pain, with fatal dose of about 50 mL.[6] On skin and eye contact, it may cause local irritation and pain.[1]

In our case, initially cardiorespiratory adverse effects

Figure 1: (a and b) Correction fluid diluter bottle displaying warning about toluene. (c) Intravenous puncture marks over the left cubital fossa with no remarkable local skin reaction at injection site

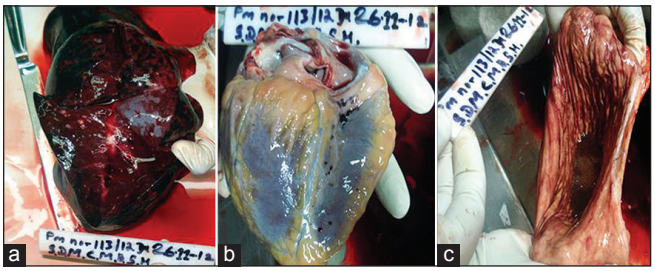

Figure 2: Necropsy findings of congested and edematous lungs (a), epicardial petechial hemorrhages over posterior surface of right ventricle (b), and gastric hemorrhages with reddish-brown blood as contents (c) in a case of suicide by intravenous injection of toluene

Table 1: Clinical features of toluene inhalation in relation to the content of toluene[6-9]

Content of toluene	Symptoms and signs
50 ppm	Slight drowsiness and headache
50-100 ppm	Irritation of the respiratory tract
100-200 ppm	Fatigue and dizziness
200-250 ppm	Similar to drunkenness, numbness, and mild nausea
>500 ppm	Mental confusion and incoordination
>10,000 ppm	Unconsciousness and death

like dyspnea, chest discomfort, tachypnea, tachycardia, and hypoxemia developed within minutes; acute pulmonary edema and pneumonitis developed within 4 h; abdominal pain, vomiting, hematemesis, and respiratory failure within 8 h; stupor by 12 h; and sudden cardiac arrest following ventricular tachycardia and death occurred 16 h after intravenous toluene. Psychological autopsy revealed underlying severe depression, inhalant addiction, and marital discord as the precipitating factor for suicide. Renal tubular acidosis as a consequence of toluene sniffing was first reported by Taher *et al*., in 1974,[10] presentation of which can mimic hypokalemic periodic paralysis.[11] Necropsy and histopathological findings supported the effects of toluene on vital organs like lungs, heart, and kidneys in our case. The mechanisms by which toluene induces renal tubular acidosis and hypophosphatemia remain to be elucidated. Further case studies on the toxic renal effects of this aromatic hydrocarbon are necessary.

Blood and urine toxicology screening during admission in suspected cases of toluene poisoning should be considered in the diagnosis of young patients presenting with unexplained hypokalemic paralysis and a normal anion gap metabolic acidosis. Toxicology screening should be an important test to establish this diagnosis. Management of toluene poisoning is supportive as no specific antidote is available and depends on the mode of contact.[1] In cases of skin or eye contact, decontamination is recommended.[6-9] In cases of inhalation, the subject should immediately be removed to fresh air to wash out the poisonous substance through the lungs. A clear airway should be ensured. Oxygen therapy and bronchodilators are also useful.[6-9] In cases of ingestion, supportive therapy is recommended and use of emetics, activated charcoal, or cathartics is contraindicated for the risk of aspiration pneumonitis.[1,6-9]

In summary, volatile solvents are commonly abused inhalants,[5] but its intravenous use as a mode of suicide is uncommon. Tissue injury mainly depends on the concentration of toluene and the duration of its presence in circulation.[3] Treatment of such cases should focus on cardiopulmonary stabilization, as volatile hydrocarbons cause major injury to the lungs; the organ being the first capillary bed encountered.[6] Early and aggressive supportive care; early intubation and controlled hyperventilation to accelerate elimination of volatile hydrocarbons; and use of defibrillator in managing sudden cardiac arrest or cardiac arrhythmias, correction of hypokalemic paralysis, and normal anion gap metabolic acidosis may be conducive to a favorable outcome with minimal residual pulmonary sequelae.

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